

REVIEW

Exposure of Americans to polybrominated diphenyl ethers

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Polybrominated diphenyl ethers, PBDEs, are a class of brominated flame retardants that, like other persistent organic pollutants (POPs), have been found in humans, wildlife, and biota worldwide. Unlike other POPs, however, the key routes of human exposure are not thought to be food and fish, but rather are from their use in household consumer products, and to the high levels of PBDEs found in house dust. The exposure of Americans to PBDEs was systematically evaluated in this study. First, exposure media data on PBDE congeners were compiled. Then, an adult intake dose was derived using exposure factors in combination with these data. The exposure pathways evaluated included food and water ingestion, inhalation, and ingestion and dermal contact to house dust. These intakes were converted to a body burden using a simple pharmacokinetic (PK) model. The predicted body burdens were compared with representative profiles of PBDEs in blood and milk. The adult intake dose of total PBDEs was estimated to be 7.7 ng/kg body weight/day, and children's estimated intakes were higher at 49.3 ng/kg/day for ages 1–5, 14.4 ng/kg/day for 6–11, and 9.1 ng/kg/day for 12–19. The much higher dose for the child age 1–5 was due to the doubling of dust ingestion from 50 to 100 mg/day. The predicted adult body burden of total PBDEs was 33.8 ng/kg lipid weight (lwt), compared to representative measurements in blood and milk at 64.0 and 93.7 ng/g lwt, respectively. Most of this apparent underprediction in total concentration was due to an underprediction of the key congener, BDE 47. The value for BDE 47 half-life in the body was identified as the variable most likely in error in this exercise. Other congener predictions compared well with measurements, suggesting general validity with the approach. An important finding from this assessment is that the food intake estimate of about 1.3 ng/kg/day (of the 7.7 ng/kg/day total) cannot explain current US body burdens; exposures to PBDEs in house dust accounted for 82% of the overall estimated intakes.

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Introduction

Polybrominated diphenyl ethers, PBDEs, are a class of brominated flame retardants that are added to plastics, polyurethane foam, textiles, and electronic equipment to reduce the likelihood of ignition and to slow the burn rate if the products do catch fire. PBDEs have a common structure of a brominated diphenyl ether molecule which may have anywhere from one to ten bromine atoms attached. Depending on the location and number of bromine atoms, there are a possible 209 PBDE compounds, each termed congeners and each being assigned a specific BDE number (note: the abbreviation PBDE will be used to denote the class of brominated flame retardants, while BDE will be used in the context of PBDE congeners). For example, there are 42 tetrabromodiphenyl ether congeners (those with four bromine atoms), but only a few of them, specifically BDE 47 and occasionally BDE 66, are found in the product formulations and in environmental or exposure media (La Guardia et al., 2006).

PBDEs have been marketed in three primary formulations, the “penta” formulation, commercially known as DE-71 and Bromkal 70–5DE, the “octa” formulation — DE-79, and the “deca” formulation — DE-83R or Saytex 102E. The formulations differ in their composition of BDE congeners. The penta formulation is dominated by penta congeners (50–62% by weight) with secondary contributions by tetra (24–38%) and hexa congeners (4–12%). The octa formulation is dominated by hepta (45%) and octa congeners (33%), with secondary contributions from hexa (12%) and nona (10%) congeners. The deca formulation is composed of essentially all BDE 209 (97–99%, with 1–3% other, mainly nona, congeners), which is the congener with all 10 bromine positions occupied. The penta and octa formulations were voluntarily withdrawn from the United States (US) marketplace by their manufacturers at the end of 2004, leaving only the deca formulation currently being marketed for use in commercial products in the US. The penta and octa formulations were banned in Europe, leaving the deca formulation also the only currently used formulation in Europe (EPA, 2006a). However, Sweden banned the use of the deca formulation in August of 2006, the ban to take effect 1 January 2007 (see: <http://www.emfacts.com/weblog/index.php?p=547>).

PBDEs have captured the attention of scientists and policymakers because levels in the environment and humans

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have increased rapidly since these chemicals came into use. The rise in PBDE concentrations in blood and breast milk in North Americans (both from the USA and Canada) throughout the 1990s into the 2000s, coupled with the fact that North American body burdens exceed those of Europeans and others by factors of 10 or more, has served to focus attention on North American exposures to PBDEs. The penta PBDE formulation has garnered the most concern because it appears to be the major contributor to current environmental and human body levels. Even with both the penta and octa formulations having been withdrawn from the US market, past use and also the possibility of debromination (loss of bromine atoms) of BDE 209 and other higher brominated congeners by photolytic or biological mechanisms to form lower brominated congeners may result in the continued presence of lower brominated congeners in the environment (EPA, 2006a).

Studies have been conducted in laboratory animals to gain a better understanding of the potential health risks of PBDEs. These studies have suggested potential concerns about liver toxicity, thyroid toxicity, developmental toxicity, and developmental neurotoxicity. These findings raise particular concerns about potential risks to children (EPA, 2006a). To date, there is no evidence of carcinogenicity of any of the PBDEs except decabromodiphenyl ether, BDE 209. In a review of toxicological studies as part of a draft Toxicological Review for EPA's Integrated Risk Information System (IRIS), EPA (2006b) has proposed that the data supports a finding of "suggestive evidence of carcinogenic potential" according to the EPA (2005) draft *Guidelines for Carcinogenic Risk Assessment*.

This paper provides an assessment of the exposure of Americans to this class of persistent organic pollutants (POPs). The literature is surveyed for environmental and exposure media concentrations, as well as body burdens as represented by blood and breast milk measurements, for the United States of America (US). Exposure media concentrations representing average or background conditions are combined with central tendency exposure intake factors to arrive at an average intake value for adults. Similar estimates are also generated for toddler ages 1–5, and for two age ranges of children, 6–11, and 12–19. Intakes by adults are converted to body burdens by use of a simple pharmacokinetic (PK) model. Predictions of adult body burdens are compared against measurements representing background conditions, and the paper closes with a set of findings from the exercise.

Methods

The literature was surveyed for measurements of BDE congeners in environmental/exposure media, in blood, and in breast milk. Representative congener concentration profiles were assigned to these media for purposes of exposure

assessment. In compiling the data, it was observed that there has not been consistency in the measurement and reporting of BDE congeners. Much of the literature has focused on congeners associated with the penta formulation: BDEs 47, 99, 100, 153, and 154. Subsequently, there is a paucity of data for the primary markers of the octa formulation, BDE 183 (which is a hepta congener), and the major congener of the deca formulation, BDE 209. BDE 183 is considered a marker for the octa formulation because it is a primary congener found in this formulation, while it is not found in the penta or deca formulations. Also, it is persistent in the environment and often found at low levels when measured. There is suggestive evidence that photolytic debromination of BDE 209 results in the formation of BDE 183 (Stapleton and Dodder, 2006), so its presence could also reflect past use of the deca formulation. As the deca formulation is the primary formulation of PBDEs marketed and used worldwide, BDE 209 is now more regularly measured, but that was not always the case. Surveys in the 1990s into the 2000s mostly did not include BDE 209, and even current surveys often do not include this critical congener. Analytical difficulties, evidence that it is rapidly eliminated from biota, and citation of previous literature focusing on the prevalence of the penta formulation congeners, are the primary reasons given for not measuring or reporting BDE 209.

So while nearly all studies include at least the five key congeners of the penta formulation, some studies report on the concentrations of over 20 congeners. Unlike dioxin-like compounds where 7 dioxins and 10 furans have been identified as toxic congeners of concern, there is no list of key toxic BDE congeners. Subsequently, there is no uniformity on which congeners should be measured, and when citing a "total" concentration (sum of all congeners) measured in a study, one must be careful to identify which congeners constitute that total.

The following congeners are the focus of this effort: 28, 47, 99, 100, 138, 153, 154, 183, and 209. In addition to the five primary congeners of the penta formulation, BDEs 183 and 209 are added as principal markers of the octa and deca formulations, respectively, and 28 (a tri BDE) and 138 (a hexa BDE) are found in the penta formulation and often also included in environmental measurements. "Total" concentrations derived in this paper will always refer to the sum of these nine congeners. However, when citing other literature, the total concentrations refer only to the congeners measured, and when pertinent, absence of the key congener, BDE 209, will be noted. Congeners which have also been reported in the literature include 17 (a tri BDE), 66 (a tetra BDE), 85 (a tetra BDE), 197 (an octa BDE), 203 (a nona BDE), and 206 (a nona BDE). The main reason they are excluded here is the paucity of data in the literature on their presence and, when measured, the levels found are often very low.

The objective of surveying the literature was to determine congener concentration profiles in exposure media for

purposes of estimating average intakes for the adult general population of the US. The “general population” is defined as the population exposed primarily to background levels that occur in the home and normal work environments; general population exposures do not include occupational exposures or exposures to special populations such as infants. In addition to adults, exposures will be estimated for three age ranges of children: 1–5, 6–11, and 12–19. Key considerations for the final assignment of concentration profiles include: (1) *the studies from which they are derived represent US conditions*. While the final selection of representative media concentrations came from US data, in some cases there is similar and maybe more comprehensive data from Canada, the United Kingdom (UK), and other countries. Often, all that is available from the US are smaller, regional, opportunistic samplings, which might not be considered representative of general populations of the US; (2) *studies with a full suite of congeners including BDEs 183 and 209 are chosen over studies without these higher brominated congeners to develop these profiles*; (3) *occupational data, while of interest, does not represent general population exposure and are not considered in this exercise*. For indoor as well as outdoor exposures, it should be clear that the data were not taken in the vicinity of known sources, such as recycling facilities, autoshredding facilities, manufacturing facilities, and the like, and (4) *studies with a complete and appropriately background data set are chosen to develop these profiles, even if they represent only one geographic region of the US*. Attempts to average congener data across studies should be done with caution, if at all. Averaging congener data across sites, when some sites did not have the full suite (and in particular, did not have BDE 209), may result in a profile not representative of background conditions.

The concentrations selected from each study will be either the standard mean (or average) or the geometric mean concentrations, whichever is provided by the author. The proper mean to use is a non-trivial issue for PBDEs, as almost all studies of PBDEs in environmental media and human tissues are characterized by very high concentrations at the high end of the distribution, such that the geometric mean is sometimes an order of magnitude or more lower than the standard mean. Most often only standard means were available, so those were used, but if geometric means were provided, they were used instead. Also, the means as provided by the authors may or may not have been derived assuming non-detects were equal to zero. If available, information on derivation of means will be provided in study summaries below.

The final selected profile of exposure media congener-specific concentrations are combined with exposure factors to estimate congener-specific intake quantities in units of ng/day, which are converted to ng/kg body weight/day, assuming body weights for the adult and three age ranges of children. The exposure factors used here were used by

EPA to estimate background exposures to dioxin-like compounds in EPA's *Dioxin Reassessment* (EPA, 2003), with one important addition, as will be described shortly. In that assessment, as here, the intent was to characterize general population exposures to background concentrations of dioxin-like compounds. All of the exposure parameters and a brief description of the pathways are provided in Table 1. Further information on these assignments can be found in EPA (2003).

The important addition to the procedures originally laid out for dioxin-like compounds, applied here to PBDEs, relates to the importance of the indoor *versus* the outdoor environment. EPA (2003) assessed exposure to dioxin using outdoor measurements exclusively. They used air concentrations from outdoor ambient air measurements for the inhalation pathway, and measurements in background soils for soil ingestion and soil dermal contact. In the case of dioxins, the primary pathways of exposure are combustion source emissions into the open environment, with subsequent accumulation in outdoor soils and, of primary importance to dioxin exposure, in the terrestrial and aquatic food chains. Subsequently, indoor air concentrations of dioxins are not known to be different than outdoor air concentrations — less if anything, and indoor dust is similarly not expected to have substantially different concentrations than outdoor soils. The same is not true for PBDEs, however. The primary cause for PBDE exposures appears to be their use in commercial products that are part of the indoor environment (computer circuitry, foam cushions, fabrics as in curtains, etc), and as noted below in the Results sections, indoor air and indoor dust concentrations of BDE congeners are often orders of magnitude higher than outdoor air and soil. Therefore, the use of outdoor measurements in air and soil does not appear appropriate for inhalation and soil/dust pathways for PBDEs. Sjodin et al. (2004a) and Stapleton et al. (2005) recognized the importance of exposure to indoor dust when estimating soil ingestion intakes in the hundreds to thousands of ng/day total BDEs for adults and children. Their intake calculations used “soil ingestion” contact rates, applying them in total to their indoor dust measurements, as though the entire contact from “soil” is, in fact, from “indoor dust”.

The approach taken here is to estimate a weighted average concentration of “dust/soil” and air, which, in theory, considers what portion of total soil ingestion/dermal contact and inhalation comes from indoor dust and indoor air. The surrogate used to estimate this portion will be “time spent indoors”. The EPA *Exposure Factors Handbook* (EPA, 1997) provides tables on hours/day spent indoors, and for adults the recommended number of hours per day is 21, which is 87.5% (or, expressed as a fraction, 0.875) of the time. Therefore, a weighted average concentration, $C(\text{avg})$, of “soil/dust” and air that will be used in the adult soil ingestion, soil dermal contact, and inhalation pathways are, $C(\text{indoor dust/indoor air}) * 0.875 + C(\text{outdoor soil/outdoor air})$.

Table 1. Exposure pathways and factors for the PBDE intake dose estimate.

Exposure factors; units	Comment; description	Adult	Ages 1–5	Ages 6–11	Ages 12–19
Body weight, kg	Used for converting ng/day to ng/kg body weight/day	70	15	30	58
Soil ingestion, mg/day	Central tendency values	50	100	50	50
Soil dermal contact, mg PBDE absorbed/day	Surface area that contacts the skin (5700 cm ² /d for adults) * amount soil adhering to skin (0.07 mg/cm ²) * fraction PBDE absorbed thru skin (0.03) * concentration in PBDE in soil (mg PBDE/mg soil); area corresponds to head, hands, forearms, lower legs	12	2.2	3.2	11
Inhalation, m ³ /day	Unpublished estimates from recent studies at 16.1 m ³ /day for adults	13.3	7.5	12	14
Fraction indoor	Children > 12 years and adults assume 21 hr/d; 19 hr/d for children	0.875	0.792	0.792	0.875
Water ingestion, l/day	Estimates from EPA (1997) still considered current	1.4	0.69	0.79	0.97
Milk ingestion, g/day	Data from USDA (1995)	175	348	357	308
Dairy ingestion, g/day	Data from USDA (1995)	55	103	88	77
Egg ingestion, g/day	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	16.8	11.25	12.3	13.9
Beef ingestion, g/day	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	49.7	21	33	48.1
Pork ingestion, g/day	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	15.4	7.2	10.5	15.7
Poultry ingestion, g/day	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	35	16.5	26.1	33.6
Other meat, g/day	Data from EPA (1997), in units of g/kg bw/d, assumes bw above; conc. assumed to be average of beef/pork/poultry	24.5	16.5	20.7	24.4
Freshwater/marine fin fish, g/day	Data developed in EPA (2003), based on EPA (2000)	11.6	3	3.8	4.5
Freshwater/marine shellfish, g/day	Data developed in EPA (2003), based on EPA (2000)	3.8	1	1.2	1.5

Note: All exposure factors and approaches developed in EPA's Dioxin Reassessment (EPA (2003), which relied on EPA's *Exposure Factors Handbook* EPA (1997) and USDA (1995).

air) * 0.125, where “C” denotes concentration. This approach simplistically assumes that exposures are proportional to time indoors *versus* outdoors, which could be incorrect for dust/soil pathways if, in fact, the actual exposures to dust/soil all were to take place outdoors. In contrast, this is a reasonable approach for inhalation, for obvious reasons. According to the *Exposure Factors Handbook* (EPA, 1997), children under 11 years of age spend 19 h indoors, so their fraction indoors will be 0.792. Children ages 12 and higher spend the adult number of 21 h indoors.

In the dermal contact pathway, an “absorption fraction” of 0.03 (3% of PBDE in soil that adhered to the skin was absorbed into the body) was assumed. For all other pathways, the dose is characterized as an “exposure” or “intake” dose, and not an “absorbed” or “delivered” dose. For all pathways, the “intakes” are the amount of contaminant that crosses the skin barrier or is taken into the respiratory and digestive systems, but not the dose “absorbed” into the body and “delivered” to the target organ. These congener-specific intake doses are converted to body burdens using a simple one-compartment, first order, PK model. Assuming that PBDEs accumulate in body lipids, which is the single compartment in the model, the equation for the change in lipid concentrations over time is:

$$\delta C_{\text{BDE}}/\delta t = (D_{\text{BDE}}(t) * \text{ABS}_{\text{BDE}})/\text{BL}(t) - k_{\text{BDE}} * C_{\text{BDE}}(t) \quad (1)$$

where C_{BDE} is the congener-specific/route-specific (CS/RS) lipid-based concentration (ng/g lwt), D_{BDE} is the CS/RS daily dose of BDE (ng/day), ABS_{BDE} is the CS/RS

absorption fraction, $\text{BL}(t)$ is the body lipid mass over time (g), and k_{BDE} is the congener-specific first-order dissipation rate in the body (day⁻¹). As presented here, k_{BDE} is assumed to be a constant, but it too could vary over time. The solution to this partial differential equation is:

$$C_{\text{BDE}}(t) = C_{\text{BDE}}(0) * e^{(-k_{\text{BDE}})t} + [(D_{\text{BDE}}(t) * \text{ABS}_{\text{BDE}})/\text{BL}(t)] * [(1 - e^{-k_{\text{BDE}}t})/k_{\text{BDE}}] \quad (2)$$

where $C_{\text{BDE}}(0)$ is the initial body burden at time 0 for the specific congener. Assuming a constant BL and a constant dose over time, steady state lipid concentration (i.e., when t approaches infinity) is easily calculated as:

$$C_{\text{BDE}} = (D_{\text{BDE}} * \text{ABS}_{\text{BDE}})/(k_{\text{BDE}} * \text{BL}) \quad (3)$$

Equation (3) will be used to predict body burden concentrations. Except for a reasonable estimate of 17,500 g body lipid reservoir (25% body fat for a 70 kg adult; EPA (1997)), all of these terms are uncertain, and of course, the assumption of steady state conditions is an inherent uncertainty with this approach.

The absorption fraction of PBDEs sorbed to soil/house dust is an obvious critical uncertainty because of the importance of the dust ingestion pathway. It is known that organic compounds sorbed to soil, or dust, are much less bioavailable than when ingested in food or inhaled. Paustenbach et al. (2006) examined the literature on the bioavailability of 2,3,7,8-TCDD, and in their monte carlo simulations on exposures to contaminated soil, they assumed that the oral absorption fraction of this compound followed a lognormal distribution with a range of 0.005–0.63 and a

mean value of 0.35. ATSDR (2004) reviewed the literature on bioavailability of PCBs in soil, and concluded that a range of 0.40–0.65 was appropriate. Paustenbach et al. (1997) commented on the fact that the bioavailability of contaminants on house dust in general is much greater than that in outdoor soil, because house dust particles are finer than soil particles. The ABS for the soil/house dust ingestion pathway for all BDE congeners was set to 0.50 in the absence of better information. McDonald (2005) assumed absorption fractions of 0.94 for BDE 47, 0.78 for BDE 99, 0.93 for BDE 100, 0.90 for BDE 153, and 0.86 for BDE 154, based on experiments with rats, and these values will be used here for the inhalation and water/food ingestion pathways. For lack of better information, 0.90 will be assumed the absorption fraction of BDEs 28, 138, 183, and 209 for these pathways. There is no additional absorption assumed for dermal contact since an absorption fraction was already included in the dose estimates for that pathway.

McDonald (2005) cited Geyer et al. (2004) to assign half-lives within the body of 3.0 years for BDE 47, 5.4 years for BDE 99, 2.9 years for BDE 100, 11.7 years for BDE 153, and 5.8 years for BDE 154, and these values will be used here. Thuresson et al. (2006) provided estimates of 0.26 year (94 days) for BDE 183 and 0.041 year (15 days) for BDE 209, and these will be used here. For lack of better information, a mid-range value of half-life, 6 years, will be assigned to congeners BDE 28 and 138. The first-order dissipation rates, k_{BDE} , are easily calculated as $0.693/\text{hl}$ (where, hl = half-life).

Results

Exposure Media Concentrations

Table 2 provides the exposure media concentrations assumed for this exercise. Each set comes from an individual study,

even though all individual studies were limited in geographic coverage. Following are brief descriptions of the data available in the literature and the data set selected for this exercise:

Water Water has been rarely sampled for PBDEs in America, and it is questionable whether available monitoring can be considered representative of drinking water. The San Francisco Estuary Regional Monitoring Program for Trace Substances sampled water, surface sediments, and bivalves (oysters, mussels, clams) in the San Francisco Estuary for 22 congeners including the nine of this assessment (Oros et al., 2005). A total of 33 water samples were taken, with total BDEs ranging from 3 to 513 pg/l. The sum of the mean concentrations of the nine congeners of this study was 135 pg/l. The only other study found measured BDEs in Washington State rivers and lakes, and in 15 samples, the range was ND — 926 pg/l, with an average of 92 pg/l, with only two samples above 100 pg/l and no detections of BDE 209 (Johnson et al., 2006).

Outdoor/Indoor Air The California Air Resources Board (CARB) established the California Ambient Dioxin Air Monitoring Network. This network, comprised of 11 sites, began sampling BDEs in 2003 in seven of 11 of the sites, four from the Bay Area and three from the South Coast (CADAMP, 2006). There were six monthly samples in 2003, and 12 monthly samples in 2004. Individual site data and site/statewide averages were available for this exercise. Twelve congeners were measured, including 17, 28, 47, 65, 88, 85, 99, 100, 153, 154, 183, and 209. BDEs 47 and 99 had similar 12-month averages in 2004, of 53 and 51 pg/m³, respectively, with 209 the next highest at 25 pg/m³. BDE 100 averaged 13 pg/m³, and others ranged from 0.04 to 4.0 pg/m³. The total average total concentration for the 18 months between 2003 and 2004 for the nine congeners was

Table 2. Exposure media concentrations of PBDEs.

Exposure Media	28	47	99	100	138	153	154	183	209	Total	Reference; n
Water, pg/l	3.3	42.7	27.6	7.2	0.3	3.9	2.9	4.4	42.3	134.6	Oros et al. (2005); n = 33
Surface soil, ng/g dwt	—	1.9	3.6	0.4	—	5.7	4.8	37.4	15.3	69.1	Offenberg et al. (2005), n = 33
Indoor dust, ng/g	ND	1857	2352	911	181	243	156	60	2394	8154	Sharp and Lunder, (2004); n = 10
Outdoor air, pg/m ³	3.0	53.0	51.0	13.0	—	3.9	4.0	1.4	25.0	154.3	CARB (2005); n = 84
Indoor air, pg/m ³	27	177	79	16	—	5	7	—	121	432	Allen et al. (2006); Webster (2006); n = 60
Shellfish, ng/g wwt	ND	3.6	1.2	0.9	ND	ND	ND	ND	ND	5.7	Oros et al. (2005); n = 17
Finfish, ng/g wwt	0.03	0.60	0.17	0.13	0.001	0.02	0.05	0.002	0.09	1.09	Schecter et al. (2006a); n = 24
Beef, ng/g wwt	0.02	0.05	0.04	0.006	0.0001	0.006	0.004	0.001	0.003	0.13	Schecter et al. (2006a); n = 3
Pork, ng/g wwt	ND	0.08	0.12	0.015	0.001	0.02	0.01	0.009	0.02	0.28	Schecter et al. (2006a); n = 6
Poultry, ng/g wwt	0.0002	0.06	0.12	0.03	0.002	0.02	0.001	0.002	0.12	0.36	Schecter et al. (2006a); n = 3
Dairy, ng/g wwt	0.0002	0.03	0.03	0.005	<0.0001	0.004	0.002	0.002	0.04	0.11	Schecter et al. (2006a); n = 15
Eggs, ng/g wwt	0.0002	0.02	0.04	0.006	0.0001	0.004	0.003	0.0001	0.01	0.08	Schecter et al. (2006a); n = 6

Note: Concentration of “other meats” used to calculated total intakes derived as the average of beef, pork, and poultry.

154 pg/m³. Data from the seven sites and the 12 months of 2004 ($n=84$ for each congener) were used to represent outdoor concentrations. Other measurements were available from the Great Lakes area (Strandberg et al., 2001; Hoh and Hites, 2005), the Gulf of Mexico (Hoh and Hites, 2005), and the Northeast (Goel et al., 2006), with measurements ranging from above 150 pg/m³ in the Chesapeake Bay (Goel et al., 2006) to under 20 pg/m³ in the remote area of Eagle Harbor, MI, USA (Strandberg et al., 2001). Measurements in Europe are generally lower, with total concentrations near or below 10 pg/m³ (Farrar et al., 2004; Jaward et al., 2004), but some measurements in China have shown to be higher than the tens to hundreds of pg/m³ of total PBDEs (Jaward et al., 2005; Chen et al., 2006).

Only two studies could be located which measured PBDEs in the US indoor environment. One sampled air in 20 urban residences in the Boston, MA, area, and the results from this study will be used here (Allen et al., 2006; Webster, 2006). Three locations per home were sampled, including the living room, bedroom, and a "personal" sample, which was taken near the breathing zone in the bedroom. Participants turned on the samplers when returning in the evening, and turned them off when leaving to work. The geometric mean of total concentrations (12 congeners measured, including seven of the nine congeners of this study; they did not measure BDEs 138 and 183) over the 20 residences were 766, 460, and 453 pg/m³ for the personal, bedroom, and living room air. BDE 47 had the highest concentration, with geometric means of 227, 158, and 145 pg/m³ for the same three locations, with BDE 209 second highest at 174, 95, and 94 pg/m³, and BDE 99 third highest at 111, 67, and 60 pg/m³. Representative congener concentrations for this exercise were determined as the average of the three location-specific geometric mean congener concentrations, and the total concentration, derived as the sum of these seven average congener-specific concentrations, was 432 pg/m³. The only other indoor measurements found were taken in a computer training facility in a public building in California by CARB (CARB, 2005). While the total average concentration for this setting was much higher at above 1500 pg/m³, BDE 209 was lower than observed in the MA homes at about 50 pg/m³. Outside the US, indoor measurements have been made in Canada (Shoeib et al., 2004; Wilford et al., 2004), UK (Hazrati and Harrad, 2005; Harrad et al., 2004, 2006), and Kuwait (Gevao et al., 2005). Generally, UK and Canadian indoor concentrations were similar to the MA homes with total concentrations in the hundreds of pg/m³, with researchers noting a ratio greater than 10 for indoor/outdoor air concentrations, but the Kuwait measurements were lower at under 100 pg/m³. Concentrations in cars have recently been measured (Harrad et al., 2006; Mandalakis et al., 2006), with concentrations in the thousands of pg/m³, and in a study from Greece (Mandalakis et al., 2006), exposure to BDEs in cars was approximately 80% of total

inhalation exposure, despite the small amount of time spent in the car.

Soil/house Dust Only one study could be found which looked at PBDEs in surface soils in the US (Offenberg et al., 2006). A total of 33 surface soil samples were taken in 15 states and measured for 30 BDE congeners. Concentrations of total BDEs averaged 103.0 ng/g dry weight (dwt), with a geometric mean concentration of 5.3 ng/g dwt. BDE 47 was detected in 31 of 33 samples averaging 1.9 ng/g dwt over the entire data set, BDE 99 was observed in 30 samples averaging 3.6 ng/g dwt, and BDE 209 was found in 24 samples, averaging 15.3 ng/g dwt. The highest concentrations were found for BDE 183, but it was only found in three samples at concentrations ranging from 121 to 562 ng/g dwt, so that the survey wide average was 37.4 ng/g dwt. The average concentrations of the nine congeners were used here, leading to a total average concentration of 69 ng/g dwt. The only other surface soil study found, measuring background soils, was reported by Hassanin et al. (2004) on a study sampling 66 surface samples from grassland and woodland areas in the UK and Norway. Soil concentrations were lower, at near 1 ng/g dwt for total BDEs.

Studies of house dust were more plentiful. Of five studies found sampling house dust for BDEs in the US, a study by the Environmental Working Group (EWG; Sharp and Lunder, 2004) included measurements of BDEs in breast milk of 20 women, but it also included measurements in house dust from the homes of 10 participants. The 10 samples were taken in nine different states ranging from California to Oregon to the District of Columbia. The overall averages of the three highest congeners, BDE 47, 99, and 209, was similar to the finding from other studies: BDE 47 was found at an average of 1847 ng/g dwt, BDE 99 at 2352 ng/g dwt, and BDE 209 at 2394 ng/g dwt. The total concentration (sum of the nine congeners) averaged 8154 ng/g dwt, which is about two orders of magnitude higher than the outdoor total soil concentration. Other US studies included one measuring 16 homes in the District of Columbia and one in Charleston, SC, with an average total concentration of 5,900 ng/g dwt (Stapleton et al., 2005), one measuring 20 homes in Boston, MA; with geometric mean concentrations from three locations (living room, bedroom, and vacuum dust) of 6332, 13,882, and 4213 ng/g dwt (Webster, 2006), one in Texas, where the median and mean from nine samples were 2507 and 12,136 ng/g dwt, respectively (Schechter et al., 2005a), and a few others where BDE 209 was not measured. Studies outside of North America tended to show significantly lower dust concentrations. Measurements of dust in the UK (Harrad et al., 2006) and Kuwait (Gevao et al., 2005) report an overall average total of 215 ng/g dwt in the UK and a geometric mean of 9 ng/g dwt in Kuwait, although neither study included BDE 209. Fabrellas et al. (2005) report sampling done by "Euroconsumers

Organization” and “CIEMAT-POPs Group”, where dust from 100 vacuum bag cleaners from Spain (34 bags), Belgium (32 bags), Portugal (22), and Italy (12) was obtained and measured for PBDEs. While they measured congener data and showed some of it in graphical form, they reported homolog group concentrations in tables, which are by definition higher than congener concentrations. Total concentrations (sum of homolog group) were still under 1,000 ng/g dwt, with Italy having the highest, at 581 ng/g dwt, followed by Portugal at 354 ng/g dwt, Spain at 238 ng/g dwt, and Belgium at 190 ng/g dwt. It is noted that BDE 209 comprised over 60% of total in all samples, with BDEs 47 and 99 the second highest contributors at well under 20% of total. A similar trend of finding high BDE 209, although even more dramatic, was noted in a recent study measuring BDE congeners in house dust and lint from the UK and Germany (Pless-Mullooli et al., 2006). The mean from nine house dust samples collected in the Northern England and Scotland was 11,325 ng/g, of which BDE 209 comprised 11,233 ng/g of that average. Lint samples from Germany showed a lower concentration, averaging 361 ng/g, but BDE 209 still dominated averaging 323 ng/g. This is different than the US data, which showed high BDE 209, but similar levels of BDEs 47 and 99 as 209.

Fish Fish were separated into “finfish” and “shellfish” for purposes of exposure assessing, with each category incorporating marine and freshwater species. A wealth of data on PBDEs in fish were available from literature worldwide, but a lot of it was fish caught in the wild for evaluation of ecological impacts, and may not be appropriately representative for human exposure. The data deemed most appropriate for exposure assessing was retail market-place surveys, although farmed fish data would also be relevant.

There were limited data on shellfish in the US; the only shellfish data available were data on clams, oysters, and mussels from the San Francisco Estuary (Oros et al., 2005), so these data were used for the profile. While measurements were made for the nine congeners, it was stated that non-detects were found for all but BDEs 47, 99, and 100. The wet weight (wwt) average total concentration was 5.7 ng/g wwt. The retail market-place data from Schecter et al. (2006a) contained data thought to be most representative for use in this exposure assessment to characterize finfish exposures, although all the samples were taken from supermarkets in one location: Dallas, Texas. A total of 24 samples including tuna, salmon, shark, trout, catfish, and herring, were taken. The top four BDE congeners found were BDE 47, averaging 0.6 ng/g wwt, BDE 99 at 0.17 ng/g wwt, BDE 100 at 0.13 ng/g wwt, and BDE 209 at 0.098 ng/g wwt. The mean concentration of BDE 183 was 0.002 ng/g wwt, and the mean total concentration over all 24 fish samples was 1.09 ng/g wwt. Other retail market surveys showed similar

results, such as one in California showing a range of 0.04–4.9 ng/g wwt in fish (Luksemburg et al., 2004), or one in Canada including 122 fish and shellfish showing a range of 0.02 ng/g (in shrimp) to 1.6 ng/g wwt (in trout; Tittlemeier et al., 2004). Other data on farmed fish (Jacobs et al., 2002; Hites et al., 2004; Shaw et al., 2005) and retail fish from abroad (Ohta et al., 2002; Pirard et al., 2005) similarly showed concentrations mostly below 10 ng/g wwt and near the mean value of 1.09 ng/g wwt measured by Schecter et al. (2006a).

There were some substantially higher measurements taken from fish in the Great Lakes, including findings by Manchester-Neesvig et al. (2001) on 21 coho and Chinook salmon samples showing an average of 80 ng/g wwt, or the temporal study by Zhu and Hites (2004), showing an average of 120 ng/g wwt for lake trout in Lakes Superior, Michigan, Huron, and Ontario. Johnson et al. (2006) sampled 63 fish (trout, perch, bass, and others) from rivers and lakes in the state of Washington, and found a mean of 35 ng/g wwt total, a median of 2.8 ng/g wwt total, and some very high individual results including one whitefish at over 1000 ng/g wwt total, with this sample having BDEs 47 and 99, each at over 400 ng/g wwt. Rayne et al. (2003) studied the temporal trend of increasing BDE concentrations in fish from the Columbia River System in southeastern British Columbia. A total of 41 whitefish samples were obtained from the period of 1992–2000. Total PBDE concentrations in whitefish obtained at two locations increased by factors of 11.8 and 6.5 during this time, going from 6.1 ng/g wwt (avg) in 1992 to 19.1 ng/g wwt in 94/95 to 71.8 ng/g wwt in 2000.

Meats/Dairy The retail market-basket survey by Schecter et al. (2006a) was also used here to assign concentrations to the remaining food types, including beef, pork, poultry, dairy, and eggs. Values they found were comparable to the other two retail market-basket surveys found for US — that from Luksemburg et al. (2004) in California and Huwe and Larsen (2005) from several states including FL, VA, CT, PA, ND, MT, OR, NM, and AZ. While the data from Huwe and Larsen (2005) appear more geographically extensive, data on BDE 209 was not included; only data from the five major penta formulation congeners (47, 99, 100, 153, and 154) were provided. Also, Huwe did not include dairy products in their survey. The total of 0.13 ng/g wwt in beef found by Schecter et al. (2006a) was derived as the average of three beef samples (two ground, one tenderloin). This is comparable to the findings in Huwe and Larsen (2005), whose beef samples ($n = 11$) averaged 0.42 ng/g lipid weight (lwt), or about 0.08 ng/g wwt (assuming 20% lipid in beef), and Luksemburg et al. (2004), whose four samples of beef averaged 0.15 ng/g wwt total. The pork total of 0.29 ng/g wwt from Schecter et al. (2006a) was derived as the average of seven pork samples (three bacon, one pork, two pork sausage, one ground pork), and this value is similarly

comparable to the results of Huwe and Larsen (2005), whose pork samples, including 11 bacon and 11 pork fat samples, showed an average total of 1.59 ng/g lwt, or about 0.24 ng/g wwt (assuming 15% lipid in pork). Schecter's total of 0.36 ng/g wwt for poultry was derived as the average of three poultry samples (chicken breast, ground chicken, ground turkey). It is comparable to the findings of Luksemburg et al. (2004): 0.41 ng/g wwt for seven poultry samples (four chicken and three turkey samples), and Huwe and Larsen (2005): 2.78 ng/g lwt or 0.42 ng/g wwt (assuming 15% lipid) in 22 chicken fat samples. Interestingly, the duck sample from Luksemburg et al. (2004) at 2.5 ng/g wwt, and the duck sample from Schecter et al. (2006a) at 1.3 ng/g wwt, were the highest of the poultry samples and these were both not included in the displayed average. This high concentration occurred because duck is very fat, listed as 75% lipid in Schecter et al. (2006a). Schecter et al. (2006a) sampled 15 dairy products, including various cheeses, cow/goat milk, yogurt, ice cream, and infant formula. The average concentration was 0.11 ng/g wwt. They also sampled six eggs, and the average concentration was 0.08 ng/g wwt, the lowest of the terrestrial food products.

Comparable concentrations of total BDEs at less than 0.3 ng/g wwt for meat products were found in Spain (Bocio et al., 2003; Gomara et al., 2006), Norway (Haug et al., 2005; Knutsen et al., 2005), and Ireland (Tlustos et al., 2005). Of particular interest is a recent survey of UK food

products published in 2006 (FSA, 2006). In that dietary survey conducted by the Food Standards Agency of the UK, meat product concentrations of BDE 209 appear exceedingly high at 3.64 ng/g wwt, and in fact, BDE 209 dominated all 19 composite food group samples, although concentrations of BDE 209 in samples other than the meat sample appear more in line with other values in the literature, at less than 0.5 ng/g wwt. For example, BDE 209 was found at 0.29 ng/g wwt in "fats and oils" and the next highest congener concentrations were BDEs 49 and 99, both reported at 0.08 ng/g wwt.

Exposure Intake Estimates

Table 3 provides the intake estimates by pathway and congener for adult exposures. The total adult estimated dose is 540.9 ng/day. The procedure was also followed for the three age ranges of children, and the resulting intakes were 740 ng/day for the 1–5 age range, 433 ng/day for the 6–11 range, and 528 ng/day for the 12–19 age range. On a body weight basis, the doses are 7.7 ng/kg/day for the adult (assuming 70 kg body weight), 49.3 ng/kg/day for ages 1–5 (15 kg), 14.4 ng/kg/day for 6–11 (30 kg), and 9.1 ng/kg/day for 12–19 (58 kg). The much higher dose for the child age 1–5 was due to the doubling of soil/dust ingestion from 50 to 100 mg/day. Other observations from Table 3 include: (1) *the pathways of soil/dust ingestion and dermal contact overwhelm the exposure of adults and children to PBDEs*. Soil/dust

Table 3. Adult exposure intakes for all congeners and pathways, and predicted body burdens compared against measured congener concentrations in blood and milk.

Description	28	47	99	100	138	153	154	183	209	Total	Fraction
I. Exposure Pathway											
	ng/day										
Water ingestion	<0.01	0.06	0.04	0.01	<0.01	0.01	<0.01	0.01	0.09	0.2	<0.01
Soil/dust ingestion	— ^a	81.3	102.9	39.9	7.9	10.7	6.9	2.9	104.8	357.3	0.66
Soil/dust dermal contact	—	19.5	24.7	9.6	1.9	2.6	1.7	0.7	25.2	85.9	0.16
Inhalation	0.3	2.2	1.0	0.2	—	0.1	0.2	<0.01	1.5	5.5	0.01
Shellfish ingestion	—	13.7	4.6	3.4	—	—	—	—	—	21.7	0.04
Finfish ingestion	0.4	7.0	2.0	1.5	0.01	0.2	0.6	0.02	1.0	12.7	0.02
Beef ingestion	1.0	2.5	2.0	0.3	<0.01	0.3	0.2	0.1	0.2	6.6	0.01
Pork ingestion	—	1.2	1.9	0.2	0.02	0.3	0.2	0.1	0.3	4.2	0.01
Poultry ingestion	0.01	2.1	4.2	1.1	0.1	0.7	0.04	0.1	4.2	12.6	0.02
Other meats	0.2	1.6	2.3	0.4	0.03	0.4	0.1	0.1	1.2	6.3	0.01
Milk ingestion	0.04	5.3	5.3	0.9	0.01	0.7	0.4	0.4	7.0	20.1	0.04
Dairy ingestion	0.01	1.7	1.7	0.3	—	0.2	0.1	0.1	2.2	6.3	0.01
Eggs ingestion	<0.01	0.3	0.7	0.1	<0.01	0.1	0.1	<0.01	0.2	1.5	<0.01
Total	2.0	138.5	153.3	57.9	10.0	16.3	10.5	4.5	147.9	540.9	
Fraction	<0.01	0.26	0.28	0.11	0.02	0.03	0.02	0.01	0.27		
II. Body burdens											
	ng/g lipid weight										
Predicted	0.3	8.6	15.6	3.3	1.1	3.7	1.1	0.02	0.1	33.8	
Blood, observed ^b	1.4	33.9	10.8	5.2	0.2	9.9	0.8	0.4	1.4	64.0	
Mother's milk, observed ^c	3.8	50.0	10.0	12.0	—	16.0	0.8	0.3	0.8	93.7	

Notes:

^a— "—" indicates no data available, or only NDs available, in which case zero concentration rather than 1/2 detection limit is used.

^bBlood data from Schecter et al. (2005b).

^cMother's milk data from NEW (2004).

ingestion and dermal contact together account for 82% of the total adult exposure. The estimates of 357 ng/day of PBDEs via soil ingestion is consistent with Sjodin et al. (2004a), who calculated an intake of 400 ng/day assuming a dust ingestion rate of 100 mg/day and comparable BDE dust concentrations as were used in this exercise; (2) *only about 17% of exposures, or 1.3 ng/kg-day, are due to food ingestion.* On the other hand, the literature has an abundance of dietary surveys of PBDEs and discussions on exposure via food consumption. The literature may be prematurely grouping PBDEs with other POPs like dioxins and PCBs, where food exposures are known to predominate. This finding that house dust dominates exposures is not unique to this study. Others have looked at other pathways and, like this assessment, have found dust to dominate all pathways at all ages (Jones-Otazo et al., 2005). Schechter et al. (2006a) focused on food intakes of PBDEs, and concluded that food intakes could not explain the high body burdens of PBDEs in Americans, and that indoor dust and air may play an important role in addition to food; (3) *the percentage of total dose attributed to BDEs 47, 99, and 209 are about equal at 26%, 28%, and 27%, respectively, followed by BDE 100 at 11%, for a total of 92% among those four congeners.* Soil-related exposures dominated for the individual congeners, but mostly for BDE 209, where soil ingestion and soil dermal contact explained 95% of the dose, while it explained 77% of exposure to BDE 47 and 88% to BDE 99. Exposures to BDEs 138, 153, and 154, were all low at between 2% and 3% of total, even though BDE 153 and 154 are almost always measured in environmental media and are considered markers for the penta and octa PBDE formulations. BDE 183, a marker for the octa PBDE formulation (or present in environmental media as a result of debromination of higher brominated BDEs such as BDE 209), was a small contributor to overall dose, at 1%. BDE 28 contributed less than 1% of total dose. However, from a body burden perspective, BDE 28 makes up between 2% and 4% of total body burden, while BDE 138, which makes up 2% of the dose, is virtually absent in body tissues. This highlights the importance of the interplay between dose and body burden when understanding and quantifying exposure to this class of compounds.

Several researchers have measured PBDEs in exposure media and estimated the exposure dose associated with that particular media. Their approach was simply to associate the media concentration with a contact rate — a given concentration in dust times an amount of dust ingested per day provides an estimate of the daily dose via dust ingestion. This was done most often for studies on house dust and food, but researchers also considered inhalation exposures when measuring air.

Stapleton et al. (2005) included the most comprehensive dust pathway estimate with their study on house dust. Using estimates of inadvertent ingestion of dust by young children (ages 1–4), 20–200 mg/day, total ingestion of PBDEs ranged

from 120 to 6,000 ng/day. They list an adult exposure of 3.3 ng/day, but this is based on a low estimate of 0.56 mg/day of dust ingestion. This value was found in EPA (1997) *Exposure Factors Handbook*, where Hawley (1985) is cited for using a value of 0.56 mg/day to characterize adult exposure to house dust from normal activities in the house (higher exposures of over 100 mg/day resulted from “work in the attic”). Sjodin et al. (2004a), in contrast, assumed an upper limit ingestion rate of 100 mg/day, and using their median concentration of 4,200 ng/g in house dust from Atlanta (range of 530–29,000 ng/g), they suggest that this pathway could add up to 400 ng/day of BDE exposure. Dust ingestion estimates outside of the US were lower because of lower dust concentrations. Using data that originated from Kuwait, Gevaio et al. (2005) used standard exposure assumptions for dust ingestion for children (100 mg/day) and adults (10 mg/day) and found that the mean ingestion of total PBDEs averaged 2.0 ng/day for children, and 0.2 for adults. Harrad et al. (2006) used indoor dust measurements from eight homes in the UK to estimate a possible range of adult dust ingestion exposures from 0.9 to 22 ng/day total BDEs, and a range for toddlers of between 12 and 43 ng/day (not including BDE 209). Jones-Otazo et al. (2005) estimate that 90% of a toddler’s total intake of 264 ng/day (not including BDE 209) originated from exposures to house dust, mainly dust ingestion. This was not only due to high levels they predicted to occur in indoor dust (through comprehensive environmental fate modeling), but also to the soil/dust ingestion rate of 50 mg/day.

Dietary dose estimates were developed mostly by individuals who also measured food concentrations. Schechter et al. (2006a) combined their measured average food concentrations with food consumption rates to calculate intakes for various age ranges (2–5, 6–11, 12–19...> = 60) and for males and females. Their results were similar to those derived here, not surprisingly because estimates here used their concentrations and food intake values were comparable. Total intakes ranged from about 0.9 to 1.5 ng/kg body weight/day for males/females above the age of 12, and their intakes for ages 2–5 was 2.7 ng/kg/day, for ages 6–11 was 1.8 ng/kg/day. Huwe and Larsen (2005) estimated a dietary intake of BDEs from meats for a consumer of “lean meats” (5% lipids) of 0.3 ng/kg/day, while a “higher fat meats” consumer had an intake of 0.8 ng/kg/day. Luksemburg et al. (2004) provided estimates assuming highest and lowest concentrations found, coupled with average adult and children ingestion rates. Their daily intakes of BDEs through fish ingestion ranged between 0.1 and 1.0 ng/kg/day in children and between 0.02 and 1.0 ng/kg/day in adults. Their intakes of BDEs in beef and chicken ranged between 0.4 and 20 ng/kg/day in children and between 0.4 and 10 ng/kg/day in adults.

Seven studies were found providing dietary dose estimates outside of the US. Bocio et al. (2003) estimated that dietary

intake equaled 97.3 ng/day for total PBDEs for adults in Spain, based on a total diet survey. Harrad et al. (2004) measured PBDEs in duplicate diet samples from both a vegan and omnivorous diet. They estimated a dietary exposure average of 107 ng/day (median = 91 ng/day) for omnivores using consumption data from the survey. Although they did not estimate an exposure intake for the vegans, they provided the omnivorous and vegan concentrations, and it is noted that the vegan concentrations were about one-half the omnivorous concentrations. Knutsen et al. (2005) combined concentrations from a market-basket survey with a comprehensive food consumption survey to estimate a mean daily adult exposure of 62.5 ng/day for Norwegians. Bakker et al. (2006) also combined food consumption data with composite food data to find a median dietary intake of 0.79 ng/kg/day for the Netherlands, with a 95% of 1.62 ng/kg/day, dominated by dairy and fish at 39% and 28%, respectively. An estimate of 51 ng/day was derived for diet only for the Swedish general adult population (Därnerud et al., 2001).

It is noted that BDE 209 was not included in any of these five European surveys; the standard suite of penta formulation BDE congeners: 47, 99, 100, 153, and 154, were included. However, three recent European survey efforts have included BDE 209. One was in Belgium, and data on the exposures was fairly low and, while not providing congener-specific concentrations, the total at 35 ng/day suggests that BDE 209 was not driving the results (Voorspoels et al., 2006). A second in Spain arrived at a similar daily total of 38.5 ng/day (Gomara et al., 2006). Congener-specific data was provided for the food products, and it was found that BDE 209 dominated egg and oil concentrations, although it was a secondary contributor to BDEs 47 and 99 in other food products, and levels were generally low. However, the UK Food Standards Agency (FSA, 2006) found BDE 209 at the highest level of all BDEs in all food products tested (as noted in the section above on food concentrations). They estimated exposure doses in conjunction with their food concentrations, and found average intakes totaling 5.9 ng/kg/day, of which 4.5 ng/kg/day was due to BDE 209. These results either are questionable themselves, or alternately, throw into question other European surveys on "total" dietary dose of BDEs, which have not measured BDE 209 in the food. It is noted that in a study on BDEs in dust in Europe, BDE 209 dominated substantially over other congeners (Fabrellas et al., 2005), providing support to this finding in food, and suggesting that much of the European literature on exposure to BDEs does not tell the complete story by not considering BDE 209.

McDonald (2005) developed a dose estimate starting from body burdens and working backward using PK modeling. He used the same model as used here, only he used it backwards. Starting with body burdens he gleaned from

several studies, he estimated individual congener intake doses of BDEs. Conducting his exercise only on the five penta formulation congeners, he estimated total doses of 8.5, 16.0, and 53.6 ng/kg/day for the median, mean, and 95% of the surveys of BDEs in blood he used in his exercise. His value of 8.5 ng/kg/day is higher than found here, at 7.7 ng/kg/day, and the estimate here also includes BDE 209. But, generally, these two results are reasonably close given that they were derived by going in different directions — by estimating intakes here in a forward manner and by McDonald's (2005) method that estimated intakes by working backwards from body burdens.

The only other estimates of dose in the literature pertain to the inhalation pathway. Researchers used indoor air concentrations and found doses reasonably similar to the 5.5 ng/day found in this exercise. Using data from passive indoor air in a Canadian study, Wilford et al. (2004) found that the median exposure via inhalation was 1.9 ng/day for females and 2.0 ng/day for males. Hazrati and Harrad (2005) used passive air measurements in 12 homes, 10 offices, and one private car to estimate a mean daily intake via inhalation of 4.3 ng/day in the UK. Harrad et al. (2006) used other air data to conclude that average inhalation intakes were 2 ng/day total PBDEs and less in the UK. Using air concentration data from a study in Kuwait, Gevaio et al. (2005) estimated inhalation doses of 0.4 ng/day for adults and 0.2 ng/day for children.

Exposure as Characterized by Body Burdens

Like other POPs, BDEs are lipophilic and body burdens are most often quantified by surveys of BDEs in blood and mother's milk. A few studies of PBDEs in adipose tissue (Petreas et al., 2003; Johnson-Restrepo et al., 2005) show lipid-based levels similar to blood and milk, and they will not be summarized here. Similar to exposure media concentrations, a single study of those available for blood and milk will be selected to be representative of background BDE in these body tissue matrices. Also like exposure media concentrations, all studies were limited geographically, so the representativeness of studies selected to represent blood and milk must be considered an uncertainty for this exercise. The concentrations in these two selected studies are provided in Table 3.

Blood The study selected to represent blood reported on BDE measurements in two-pooled samples of 100 individual each, and 39 samples of blood from individuals in Mississippi and New York (Schecter et al., 2005b). Also, the study included an archived blood sample from 1973, which was from a pool of 100 individuals from Dallas, Texas. All samples were analyzed for 13 BDEs including 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209. All congeners were non-detects for the 1973 sample. The two-pooled samples collected in 2003, one serum and one whole blood,

were from the University of Texas Southwestern Medical Center in Dallas. The 39 individuals sampled in 2003 included 29 from Mississippi and 10 from New York. The results from the three groups of samples from 2003 were fairly similar; total concentrations of the 13 congeners were 61.8 ng/g lwt for the serum pool, 79.7 ng/g lwt for the whole blood pool, and 52.6 ng/g lwt as the mean for the 39 individuals. The congener-specific trends were similar as well, with BDE 47 dominating the profile at between 44% and 53% of the total, with BDE 99 and 153 comprising about the same amounts — BDE 99 was between 14% and 21% and 153 was between 11% and 20%. From the individual samples, an interesting trend was that women had higher concentrations than men: the range and mean in 22 men were 4.6–192.8 ng/g lwt and 25.1 ng/g lwt, and for 17 women the range and mean were 5.6–365.5 ng/g lwt and 74.1 ng/g lwt. BDE 209 was found at low levels in the pooled blood, 1.4 ng/g lwt, and in the individual samples, averaging 1.7 ng/g lwt with non-detects in 19 of 39 individual samples. The total concentration of the nine congeners of this study, with congener-specific concentrations derived as the average of the two-pooled and one average of the 39 individual samples, was 64 ng/g lwt.

Several additional studies on blood measurements from the US reported similar concentrations. Schechter et al. (2006b) report on a different study measuring the concentrations of 12 BDEs, including BDE 209, in the blood of eight vegans. They found a range of BDE concentrations of 12.4 to 127 ng/g lwt total, with a median of 23.9 and a mean of 53.3 ng/g lwt. The congener found highest was 47, with a mean concentration of 23 ng/g lwt. Sjodin et al. (2004b) conducted a rigorous temporal evaluation collecting samples representing different time frames from the mid-1980s until the early 2000s, and found total concentrations rising from 9.6 ng/g lwt in the 1985–1989 time frame, to 48 ng/g lwt in 1990–1994, 71 ng/g lwt in 1995–1999, and 61 ng/g lwt in 2000–2002. Ninety-three anglers from New York and New Jersey who completed a fish consumption questionnaire were sampled between 2001 and 2003 (Morland et al., 2005). The highest congener found was BDE 47, at a geometric mean of 13.3 ng/g lwt, followed by BDE 99, at 3.2 ng/g lwt, BDE 153 similarly at 3.2 ng/g lwt, and BDE 100 at 2.7 ng/g lwt. BDE 209 was not reported, and all other congeners were not detected or very infrequently detected with geometric means less than 1 ng/g lwt. There were moderate but statistically insignificant increases in BDE concentrations as one went from no local fish intake to >1 meal/week. Twelve paired samples of maternal and cord blood were obtained from a hospital in Indianapolis during August–December, 2001, and analyzed for BDEs 47, 99, 100, 153, 154, and 183 (Mazdai et al., 2003). Results for maternal and cord blood were essentially identical: the range and median of total PBDE for mother's blood was 15–480 ng/g lwt and 37 ng/g lwt, and the corresponding range and median for infant

blood was 14–460 ng/g lwt and 39 ng/g lwt. Wolff et al. (2005) conducted a study of exposures among mothers who were pregnant near the World Trade Center Site on 11 September 2001. The study involved a complex evaluations of exposures, including measurement of key persistent contaminants including polycyclic aromatic hydrocarbons, polychlorinated biphenyls, dioxins, and PBDEs. The authors did not find an association of PBDEs and measures of potential exposure to WTC contaminants, and generally found low levels of BDEs. Of 100 mothers, they found median serum levels for BDEs 28, 47, 99, 100, and 153 at 0.65, 9.7, 1.5, 1.8, and 1.8 ng/g lwt respectively.

The most interesting recent study of BDEs in blood was a case study of a single family in California (Fischer et al., 2006). The results indicated somewhat elevated levels in the parents, but higher levels in one child and still higher levels in the toddler of the family. Samples were collected from a family of four, including 35 and 37 year-old parents, a 5-year-old daughter, and an 18-month-old son in September and December of 2004. The sum of BDEs 47, 99, 100, 153, and 154 in the parents ranged between 64 and 147 ng/g lwt in the two sampling dates, and BDE 209 contributed a relatively small addition at between 2 and 23 ng/g lwt. The story was much different with the children. The 5-year-old had concentrations of 237 and 244 ng/g lwt in the two sampling dates for the five penta formulation congeners (244 ng/g lwt was the average of duplicate sampling of 239 and 249 ng/g lwt), but a disparate range of 143 ng/g lwt of BDE 209 in the September sample and 11 ng/g lwt in the December sample (again average of duplicates in December). The toddler had the highest concentrations of all: 418 and 482 for the five congeners and 233 and 23 ng/g lwt of BDE 209 in the September and December samples, respectively. Discounting laboratory error, the authors attribute the higher concentrations in the children to exposure to house dust; elevated levels of BDE 209 support this hypothesis because studies have shown elevated levels of BDE 209 in dust, as discussed above. The authors attribute the drop in BDE 209 between the sampling dates to a combination of a rapid half-life of this congener in humans, combined with reduced exposure between the two sampling dates.

In summary, these data suggest a typical range of ~30–80 ng/g lwt of total BDEs as representative of blood in the general population of Americans in the 00s, with some measurements as high as the hundreds of ng/g lwt. In contrast, most studies from Europe, Asia, and elsewhere suggest concentrations of total BDEs in blood less than 10 ng/g lwt.

Blood from 37 Swedish men was sampled in 1991 and 2001, and measured for 15 congeners including BDE 209 (Jakobsson et al., 2005). An additional 10 men were sampled in 1988 and 2002. These men were specifically selected to represent those who consumed high amounts of fish, so they do not necessarily represent a cross-section of the average

population. The median and range of total concentrations from the 37 men from 1991 and 2001 was 11 (3.3–59) and 14 (4.2–57), expressed in units of pmol/g lwt. Because individual results were not provided, these totals could not be converted to ng/g lwt, but the total would be lower than these values in units of ng/g lwt, since individual congener conversion factors range from about 0.5 to 0.9. Pooled samples of about 20 individuals each from five hospitals in Norway (total number of samples analyzed was not provided) were analyzed for 11 BDEs, not including BDE 209. Samples for the years 1977, 82, 88, 91, 94, 97, 98, 99, 2000, 01, 02, and 03 were obtained. The sum of the seven most abundant congeners showed a concentration range of 0.5–5.0 ng/g lwt, with a clear trend of low concentrations for the early years — 0.5 in 1977, 1.3 in 1982, etc., rising to consistently higher levels (but still low compared to US levels) between 3.6 to about 5.5 ng/g lwt between 1997 and 2003. Blood concentrations of pregnant Faroese (the Faroe Islands are between Shetland and Iceland, and Faroese depend on marine fish and mammals for food) women were determined from samples taken in 1994, and their children's blood was sampled and measured 7 years later in 2002 (Fangstrom et al., 2005). Fifty-seven mothers and 42 children were sampled, of which 41 were mother/child pairs. BDEs 47, 99, 100, 153, 209, and 153/154 were measured. Concentrations were low, with a median total concentration of just over 5 ng/g lwt for both mothers and children. The dominant congener for the mothers was 47 and the co-eluting 153/154, explaining about 26% each, while for the children, the predominant congener was 153, explaining about 46% of the total concentration. BDE 209 was present in two mothers at low concentrations of 0.8 and 1.0 ng/g lwt, respectively. Samples of maternal blood plasma, cord blood plasma, and breast milk were taken in 2000–2001 from 15 mothers living in Stockholm (Guvenuis et al., 2003). The median and range from the three matrices, in ng/g lwt total (11 congeners but not including BDE 209), were: maternal blood — 2.1 (0.7 — 8.4); cord blood — 1.7 (0.5–4.3); breast milk — 2.1 (0.6–7.7).

The only study that could be found which presented blood data outside of the US with concentrations comparable to those found in the US was a study in Nicaragua (Faldt et al., 2005). Five pools of serum from teenagers who lived and/or worked near a waste disposal in Managua, Nicaragua, and four pools of serum from women in different settings (urban areas, fishing villages, etc.) were analyzed (one analysis per pool) for BDEs 47, 99, 100, 153, 183, 203, and 209. The pool of teenagers who both worked at the disposal site and lived nearby (no other pool was that exposed) had the highest concentrations, with a total over 600 ng/g lwt. The average of the other eight pools was 38 ng/g lwt. BDE 47 was the most prominent congener, contributing just under 50% of the total concentration, with BDE 99 second at about 20%, and BDE 100 third at 11%. BDE 209 was present at equal levels in the

teenagers living near and working at the disposal site, as compared to all other groups, at about 5 ng/g lwt.

Mother's Milk While blood data suggested concentrations of total BDEs in the range of 30–80 ng/g lwt, data on BDEs in breast milk suggest possibly higher concentrations, with medians or means in some studies in the US above 100 ng/g lwt. The study selected to be representative was conducted by the Northwest Environment Watch in 2003 (NEW, 2004). Between April and November of 2003, 40 first-time breastfeeding mothers from the Pacific Northwest, 10 each from Washington, Oregon, British Columbia, and Montana, were sampled for the presence of 32, 28/32, 47, 66, 71, 85, 99, 100, 153, 154, 183, and 209. Total concentrations ranged from 6 to 321 ng/g lwt in the breast milk, with median and mean levels of 50 and 97 ng/g lwt, respectively. BDE 47 was the highest found, with a mean level of 50 ng/g lwt, followed by 153 at 16 ng/g lwt, 100 at 12 ng/g lwt, and 99 at 10 ng/g lwt. BDE 209 was found in 24 of 40 samples, with a high of 4 ng/g lwt and a mean of 0.8 ng/g lwt. The mean concentration of the nine congeners of this study was 93.7 ng/g lwt.

Several additional studies from the US and Canada provided comparable results. A similar study was conducted by the Environmental Working Group (EWG; Lunder and Sharp, 2004) and might have been chosen as the representative study, except that levels found were among the highest found in the US. The EWG sampled 20 primiparae women from around the country for 35 BDEs in all. Total BDEs averaged 159 ng/g lwt, ranging from 9.5 to 1,078 ng/g lwt, with six having levels above 100 ng/g lwt, and two exceeding 700 ng/g lwt. Schecter et al. (2003) collected milk from 47 volunteer donors between August and December 2002; 24 donors were from Austin, TX, USA; and 23 were from Dallas, TX. Samples were measured for 13 BDEs, and the mean total BDE concentration was 74 ng/g lwt (median = 34; max = 419). BDE 209 was quantified in only seven samples, with 16 NDs and the rest no measurements. Focant et al. (2004) evaluated two different analytical methods on both blood and milk samples, and found comparable results for both methods. They analyzed three pooled samples of mother's milk: one pool from two mothers in Denver, one pool from 10 samples collected in 2003 in California, and the third pool from 10 individuals in North Carolina also in 2003. The average total BDE from these three was 315 ng/g lwt, with BDE 47 the highest at 193 ng/g lwt (61% of total), followed by BDE 99 at 55 ng/g lwt (17%) and 100 at 34 ng/g lwt (11%). Breast milk was collected from 46 women in the Boston, MA, USA; area, with total BDE concentrations ranging from 4 to 263, with a geometric mean of 28 ng/g lwt (Wu et al., 2005). Only one sample, the highest at 263 ng/g lwt, was higher than about 130 ng/g lwt. A limited set of longitudinal data (i.e., data on changes over time) was available for three women for BDEs 47 and 99

(Sjodin et al., 2005). Samples were taken at different days during a single lactation. Contrary to expectations, levels of these contaminants increased over time in nearly all cases. BDE 47 increased from 30 to 40 ng/g lwt in two of three women from postpartum day 40 to 120, and from 10 to about 15 ng/g lwt from days 40 to 60 in the other woman. BDE 99 increased from 50 to 100 ng/g lwt in one participant from days 40 to 120, increased slightly from maybe 5 to 6 ng/g lwt from days 40 to 60 in another, and decreased from 8 to about 5 ng/g lwt from days 40 to 90 in the third participant. This increase in concentration could be owing to a decrease in body weight leading to lower lipid reservoirs in the milk supply of the mother. With constant lipid content in milk, the normal expectation is that the lipophilic BDEs would decrease over the course of lactation, as this process represents a significant depletion mechanism in the woman. Ryan et al. (2006) reports on measurements of PBDEs in mother's milk from 2002 ($n = 14$), 2003 ($n = 13$), and 2004 ($n = 34$) from Ontario, Canada, and finds concentrations similar to that in the US. Specifically, median and mean concentrations of total PBDEs for the three years equals: 33 (median) and 139 (mean) ng/g lwt in 2002, 39 and 126 ng/g lwt in 2003, and 20 and 48 ng/g lwt for 2004, (the article did not indicate whether BDE 209 was included in these totals).

Like the blood data, the mother's milk data suggests much higher concentrations in North America compared to European and Asian countries. Extracted milk fat from the third round of the WHO-coordinated exposure study was evaluated for the presence of BDEs (Kotz et al., 2005). Samples from 17 different locations (of 24 total) were available. The highest level by far was the level found in a sample from the US at 373.6 ng/g lwt, with the second highest being 10.3 ng/g lwt from Ireland (BDE 209 not included). Thomsen et al. (2005) sampled breast milk of 151 women representing the northern, southwestern, and eastern parts of Norway. The sum of the seven most abundant congeners (BDE 209 not included) ranged from 0.95 to 21.05 ng/g lwt, with a median of 2.35 ng/g lipids. A total of 89 lactating mothers in four towns in Japan provided both serum and milk samples for analysis of 13 BDEs, including BDE 209 (Inoue et al., 2006). The geometric means for total BDEs in human milk and serum was 1.56 and 2.89 ng/g lipid, respectively. BDE 209 was the predominant congener in serum, accounting for 38% of the total amount of BDEs, but it was a minor component in milk, accounting for 8%. Fangstrom et al. (2006) conducted a temporal study on Swedish mother's milk. Fourteen pooled milk samples representing the years 1980 (116 mothers pooled), 1984/1985 (102 mothers), several of the years between 1988 and 2002 (20 mothers), 2003 (15 mothers), and 2004 (20 mothers) were sampled for BDEs 47, 77, 99, 100, 153, and 209. It was not possible to quantify 209 in milk, and the authors suggest this could be due to the short half-life of this compound that has been noted for serum. From the middle

of the 1990s, the concentrations of the lower brominated BDE congeners, 47, 99, and 100, have been decreasing, while 153 appears to be retaining its levels reached towards the latter 1990s. Overall, concentrations of individual congeners were near 1.0 ng/g lwt, and total concentrations therefore were all under 10 ng/g lwt. Ohta et al. (2002) determined the concentration of BDEs in breast milk of 12 primiparae women at 1 month after delivery in Japan. Concentrations ranged between 0.7 and 2.8 ng/g lwt (BDE 209 not included).

Converting Intake Dose to Body Burden

The congener-specific intakes provided in Table 3 were converted to body burdens using the simple one-compartment PK model described in the Methods section above. Predicted congener-specific lipid weight concentrations are also provided in Table 3, alongside the selections of the blood and mother's milk representative concentrations.

Overall, the total concentration was predicted at 33.8 ng/g lwt, lower than the observed blood concentration at 64.0 ng/g lwt and the milk concentration of 93.7 ng/g lwt. Predictions appear reasonably close to measurements for eight of nine congeners. The prediction of BDE 47 at 8.6 ng/g lwt did not match the observed measurements of 33.9 ng/g lwt in blood and 50.0 ng/g lwt in milk. The predictions of the other three congeners found most frequently were reasonable, as compared to observations: BDE 99 predicted at 15.6 ng/g lwt while it was found at 10.8 and 10.0 ng/g lwt (blood and milk, respectively), BDE 100 predicted at 3.3 ng/g lwt while it was found at 5.2 and 12.0 ng/g lwt, and BDE 153 predicted at 3.7 ng/g lwt, while it was found at 9.9 and 16.0 ng/g lwt. Excluding BDE 47, total PBDE was predicted at 25.2 ng/g lwt while it was found at 30.1 ng/g lwt in blood and 43.7 ng/g lwt in milk.

The cause for the underprediction of BDE 47 is not known, but it could very easily be the assumed half-life in humans. It is interesting to note that the predicted intakes of BDE 47 and 99 were very similar, at 139 ng/day (BDE 47) and 153 ng/day (BDE 99), but the BDE 47 concentration in lipids has been measured to be 3–5 times higher than that of BDE 99 (see Table 3). It is unlikely that the dose calculations were significantly in error, as exposure media concentration surveys were fairly consistent in the literature. This would suggest that the PK assumptions are questionable. Both are assumed to be absorbed from dust (absorption at 50% of intake for both) and food (94% for BDE 47 and 78% for BDE 99) similarly, and these values seem reasonable. The likely parameter to be assigned incorrectly is the overall rate of dissipation of BDE 47 in humans. It was assigned a value of 0.23 year^{-1} , corresponding to a half of 3.0 years. At this value, it dissipated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years. Had it dissipated at a half-life of 10 years, the prediction would jump to 28.7 ng/g lwt, about two times as high as BDE 99, and more in line with measurements of BDE 47 in blood and mother's milk. Total

predicted concentration would rise to 53.4 ng/g lipid, also closer to observations.

In the single study in the literature providing human half-lives for the lower brominated congeners including BDE 47, Geyer et al. (2004), in fact, developed them based on rat data extrapolated to humans. Expectations that the behavior of these compounds in rats correlate to humans is uncertain; even the behavior of these compounds between rats and mice differ. Orn and Klasson-Wehler (1998) found that BDE 47 was persistent in the rat, but rapidly excreted in urine in mice. In a recent study, Sanders et al. (2006) found that BDE 99 was the most rapidly metabolized congener of BDEs 47, 99, and 153 following single and multiple doses of radiolabelled mixtures of these three congeners administered by gavage to rodents (rats and mice). This contradicts the extrapolation by Geyer et al. (2004) which resulted in a more rapid half-life for BDE 47. In contrast, however, Staskal et al. (2006) found that BDE 47 was the most rapidly excreted congener of BDEs 47, 99, 100, and 153 applied intravenously to female mice. Geyer et al. (2004) estimated a second set of half-lives using human data from a Swedish cohort, but provided few details on the exercise, and in fact, used food measurements to estimate their only presumed intake, diet, in their exercise. Interestingly, BDE 47 was still estimated to have a more rapid half-life, 1.8 years, by this second method, compared to BDE 99, at 2.9 years. The exercise in this paper would certainly suggest that BDE 47 is more persistent in humans than BDE 99, as the intake doses of BDEs 47 and 99 were virtually the same, at 139 and 153 ng/day, respectively, while the observed concentrations in blood and mother's milk suggest that BDE 47 bioaccumulates at 3–5 times higher levels than BDE 99 in the human body. In any case, given uncertainties in other quantities in this exercise, not to mention use of a simple one-compartment PK model, it would be premature to use this framework to “calibrate” congener-specific half-lives in humans. Still, it gives an indication of where key information gaps are, and half-life in humans is obviously critical.

Summary of findings

This assessment has entailed a compilation of literature data on PBDE exposure media concentrations, and in human blood and mother's milk. Intake dose estimates were developed using the media concentrations in conjunction with exposure factors. These intakes were converted to body burdens using a simple, one-compartment model, and then these body burdens were compared to observations. Overall findings and comments from this exercise include:

(1) The compilation of exposure media data focused on data from the US, but also summarized key studies from Europe and abroad. Generally, levels in soil, house dust, and air tended to be higher in the US. Total BDEs (sum of BDEs

28, 47, 99, 100, 138, 153, 154, 183, and 209) in these US media were in the range of (1) 100 pg/m³ outdoors but higher at 500 pg/m³ or more (depending on the proximity of sampling to a source such as a computer) indoors; and (2) under 100 ng/g dw in outdoor soil but in the thousands to tens of thousands ng/g dw in indoor house dust. Levels in food appeared comparable in the US and abroad, with concentrations of individual terrestrial animal food products generally near 0.30 ng/g ww or lower, and market basket or farmed fish at levels between 1 and 5 ng/g ww, although some fish caught in the Great Lakes and other natural settings were much higher, into the tens and even hundreds of ng/g ww. However, there was an important finding which bears further examination, and that is, in the most recent UK food survey published in 2006, BDE 209 dominated all composite food types sampled with measurements as high as 3.6 ng/g ww in meat products, while being measured at 0.1–0.3 ng/g ww in other animal food products. In contrast, BDE 209 averaged near or under 0.1 ng/g ww in all US animal food products. BDE 209 had not been measured in any other European survey found, and so given the high concentrations found, further investigation is warranted.

(2) The compilation of body burden data (as measured by concentrations in blood and mother's milk) also focused on data from the US, but again summaries of key studies abroad, mostly from Europe, were provided. It was found that body burdens of Americans are higher than body burdens of Europeans, by upwards of an order of magnitude and more. Data suggest total BDE body burdens in the range of 30–>100 ng/g lw in Americans, while it was under 10 ng/g lw elsewhere. Breast milk concentrations tended to be higher than blood concentrations, and also, there was one blood study which separated data from males and females, and the females tended to have higher blood concentrations. Generally, the predominant congener found in blood and milk is BDE 47, explaining about 50% of the total concentration. The second most abundant congeners are 99 and 153, both explaining in the range of 10–20% of total concentrations. When BDE 209 was measured, it was found in about half the samples at low levels near 1–2 ng/g lw. There was only one study providing blood data for children, and it provided data for four family individuals: two parents and two children — a toddler at 18 months and a 5-year old. The body burdens of these children were significantly higher than their parents, in the 200–400 ng/g lw range, while their parents were near 100 ng/g lw, and there was very high measurements of BDE 209 in the children, >100 ng/g lw, also contrasting blood and milk data in the general literature showing low concentrations, near 1 ng/g lw. Exposures to mother's milk for the toddler, as well as earlier lactational exposure to the 5-year-old, and greater exposures to house dust were given as reasons for these high measurements in the children.

(3) Intake estimates in the literature have tended to focus on either a dust or a food pathway; rarely on total intakes from both pathways. Similar to results here, some estimates of exposure via house dust were high, up to 400 ng/day (443 ng/day here including ingestion and dermal contact), but other estimates were as low as 3 ng/day. This low estimate assumed less than 1 mg/day dust ingestion for adults while higher estimates assume more conservative 50–100 mg/day adult dust ingestion. Dust ingestion estimates for children tended to be higher because of higher exposure to dust. Estimates of intakes from food ingestion were in the range of 0.5–2.0 ng/kg/day for both the US and Europe. As noted above in the summary on exposure media concentrations, a recent UK food survey measuring several BDEs including 209 found very high levels of this congener. The study also calculated intakes from food, and estimated an average total intake from food to be 5.9 ng/kg/day, of which BDE 209 alone was 4.5 ng/kg/day. Intake estimates of this assessment were in the range of 433–740 ng/day for children and adults, with the highest dose for the child age 1–5 owing to an increase in dust ingestion from 50 to 100 mg. On a body weight basis, the doses are 7.7 ng/kg/day for the adult (assuming 70 kg body weight), 49.3 ng/kg/day for ages 1–5 (15 kg), 14.4 ng/kg/day for 6–11 (30 kg), and 9.1 ng/kg/day for 12–19 (58 kg). These intakes were driven by indoor house dust exposures via ingestion and dermal contact; those two pathways accounted for about 82% of total intakes, with inhalation and food/water ingestion explaining the remaining 18%. On a congener-specific basis, intakes for BDEs 47, 99, and 209 were about equal at 27% of total each.

(4) Using a simple PK model parameterized with available literature values, adult lipid-based concentrations (not specific to blood or milk) were predicted, starting with these intake values. On a total PBDE basis, the prediction was low at 33.8 ng/g lwt, while it was observed at 64.0 ng/g lwt in blood and 93.7 ng/g lwt in milk in studies selected as representative of the general population. However, predictions were reasonably close to measurements for eight of nine congeners. The prediction of BDE 47 at 8.6 ng/g lwt did not match the observed measurements of 33.9 ng/g lwt in blood and 50.0 ng/g lwt in milk. The cause for the underprediction of BDE 47 is not known, but it could very easily be the assumed half-life in humans. At 3.0 years, it dissipated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years.

(5) The reasonable match of eight of nine congeners in this simple PK exercise lends credibility to the overall approach of estimating BDE intakes from exposure media concentrations and contact rates, followed by use of the simple PK model to predict body lipid concentrations of BDEs. Nonetheless, uncertainties exist throughout this exercise, including, but not limited to: the representativeness of exposure media and body burden concentrations, key exposure parameters for the dust pathways including dust ingestion rates and dermal

absorption, absorption of BDEs from ingested dust, and PK half-lives (particularly for BDE 47). Perhaps the most important finding of this work is that food and inhalation exposures do not appear to explain the body burdens of Americans. Contact rates for food/water ingestion and inhalation are fairly well established, and the exposure media concentration summaries suggest similarities among different studies. Therefore, the dose via food/water ingestion and inhalation might be considered reasonably certain, for purposes of this discussion. However, using the PK model, they explained less than 20% of the body burden. It was assumed that the remainder of the exposures came from house dust through the pathways of ingestion and dermal contact. Circumstantial evidence supporting this hypothesis was the high concentrations found in US house dust, and other researchers have also identified house dust as a key matrix of exposure concern for these compounds. Interestingly, house dust concentrations in European studies were found to be lower than in the US, by an order of magnitude and more, and Harrad et al. (2006) hypothesize that the difference in European and US body burdens (European body burdens are much lower than US body burdens) is due to exposure of Americans to high concentrations of BDEs in house dust. Still, there was no “proof” that contact with house dust explains the majority of body burdens of Americans. Indeed, some researchers assumed much lower rates of dust ingestion (Stapleton et al., 2005) as compared to this assessment leading to much smaller estimates of dust ingestion exposure. Still, the overall weight-of-evidence of this exercise supports the finding that the bulk of US exposures occur in the indoor environment through contact with house dust. The exercise suggests these exposures account for between 80 and 90% of total exposures, with the remainder due primarily to food ingestion. Nonetheless, more research is recommended to verify these findings and better quantify the uncertainties that have been identified.

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